Lesions affecting the oculomotor nucleus are rare tending to produce bilateral effects owing to the subnuclei arrangement. Our cases developed bilateral, symmetrical ptosis indicating damage to the single, midline subnucleus serving LPS. Our first case showed restriction of both up- and downgaze, indicating damage to both the superior and inferior rectus nuclei. Unfortunately, Dolls eye movements were not performed but would be expected to overcome this restriction in the short term. The absence of convergence can be attributed to damage to the superior colliculus and its connections to the lateral suprasylvian area of the parieto-occipital cortex.[7] Our second case is owing to damage of both the LPS and IV nuclei, indicating an area of damage on the posterior aspect of the nuclei arrangement.

These two cases illustrate the clinical correlation between anatomy and function in a very complicated area of human physiology. Given the unfortunate symptoms experienced by our patients, it is relatively straightforward to explain why such a treatment received for their brain tumours has led to gross problems with their ocular motility. The fact that two patients have complained of such problems within a short period of time must indicate that, although this has not been reported previously, these side effects do occur. Patients must be made aware of the possibility of their occurrence in order to give fully informed consent.

References

1 Warwick R. Oculomotor organisation. *Ann R Coll Surg Engl* 1956; **19**(1): 36–52.
2 Kandzari SJ, Belis JA, Kim JC, Gnepp DR, Riley RS. Clinical results of early prostatic cancer treated by pelvis lymphadenectomy and ^{125}iodine implants. *J Urol* 1982; **127**(5): 923–927.
3 Wilson JW, Morales A, Bruce AW, Froud P. Interstitial radiotherapy for localized carcinoma of the prostate. *Can J Surg* 1983; **26**(4): 363–365.
4 Kreth FW, Faist M, Rossner R, Birg W, Volk B, Ostertag CB. The risk of interstitial radiotherapy of low grade gliomas. *Radiother Oncol* 1997; **43**(3): 253–260.
5 Wowra B, Schmitt HP, Sturm V. Incidence of late radiation necrosis with transient mass effect after interstitial low dose rate radiotherapy for cerebral gliomas. *Acta Neurochir (Wien)* 1989; **99**(3 and 4): 104–108.
6 Obasi PC, Barnett GH, Suh JH. Brachytherapy for intracranial meningioma using a permanently implanted iodine-125 seed. *Stereotact Funct Neurosurg* 2002; **79**(1): 33–43.
7 Bando T, Hara N, Takagi M, Yamamoto K, Toda H. Roles of the lateral suprasylvian cortex in convergence eye movements in cats. *Prog BrainRes* 1996; **112**: 143–156.

JM Durnian[1], V Wong[2], J Ashworth[1], C Mallucci[3] and IB Marsh[1]

[1]Department of Ophthalmology, Walton Hospital, University Hospital Aintree, Liverpool, UK

[2]Department of Orthoptics, University Hospital Aintree, Liverpool, UK

[3]Department of Neurosurgery, Walton Centre for Neurology and Neurosurgery, Liverpool, UK

Correspondence: JM Durnian, Department of Ophthalmology, Walton Hospital, University Hospital Aintree, Rice Lane, Liverpool L9 1AE, UK
Tel: +44 151 525 5980;
Fax: +44 151 706 2000.
E-mail: jon_durnian@hotmail.com

This was presented as a poster at the 30th Meeting of the European Strabismological Association, Killarney, June 2005

Eye (2006) **20,** 1479–1481. doi:10.1038/sj.eye.6702369; published online 5 May 2006

Sir,

Giant fornix syndrome: a recently described cause of chronic purulent conjunctivitis and severe ocular surface inflammation, with a new diagnostic sign on CT

Case report

A 75-year-old lady initially presented with a red, sticky right eye for several weeks with copious yellow discharge. She had no prior ophthalmic history. Her medical history included rheumatic fever resulting in mixed mitral valve disease and pulmonary hypertension.

At presentation, she was noted to have a small peripheral corneal epithelial defect with mild infiltrate and was diagnosed as having blepharoconjunctivitis and marginal keratitis and treated with guttae chloramphenicol and topical steroid (maxidex). Lid hygiene was also advised. A conjunctival bacterial swab grew *Staphylococcus aureus*, sensitive to, among others, chloramphenicol.

One week later, the epithelial defect had improved but there had been little improvement in the discharge and she was kept under outpatient review as she suffered recurrent episodes of purulent discharge. Repeated cultures grew *S. aureus* with identical sensitivities. The absence of a mucocele was noted and syringing and

probing revealed patent flow through the nasolacrimal duct. Her left eye remained unaffected.

Fourteen months after her initial presentation, she returned acutely complaining of increased discharge and unusually severe pain and photophobia. Her right eyelids were matted together and upon cleaning there was found to be a large volume of yellow discharge emanating from beneath the upper lid, which required irrigation and furthermore, hampered the remaining examination. The eye was injected and there were signs of early corneal thinning nasally. A diffuse epitheliopathy and areas of limbal vascularisation were also present. Again, there was no mucocele. An urgent Gram stain of a conjunctival swab showed Gram-positive cocci (and later grew sensitive *S. aureus*) and on microbiological advice she was recommenced on guttae chloramphenicol.

On review 3 days later, there had been an improvement in the discharge, but the right eye remained very injected and a corneal epithelial/anterior stromal defect was evident, with no infiltrate. Despite intensive topical and oral antibiotic treatment, the nasal corneal thinning progressed with descemetocele formation (Figure 1) and signs of sclerokeratitis. Investigations at that stage revealed an ESR 56 and CRP 13.3, Rheumatoid Factor 87.9 (<20) and a positive ANA 1:40. ANCA were negative.

She was commenced on 500 mg oral methylprednisolone for 3 days, followed by a reducing dose of daily oral prednisolone. Medical and rheumatological review reported no signs of active rheumatoid arthritis or evidence for a systemic vasculitis. Her right eye settled over the following week with no further progression in corneal thinning and the epithelial defect healed.

In view of the intermittent recurrence of discharge a CT scan of her orbits and paranasal sinuses was performed. There was no sign of foreign body or frontal sinusitis. However, a pocket of air was noted deep in the upper fornix of the right eye (Figure 2). On subsequent examination, our patient was noted to have age-related dehiscence of the levator aponeurosis (Figure 3) and a diagnosis of Giant fornix syndrome was made. Her eye remains quiet with occasional upper fornix irrigation and on maintenance topical steroid and antibiotic therapy.

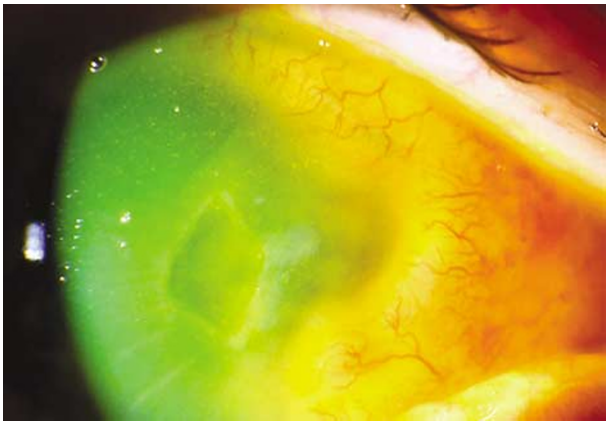

Figure 1 Severe ocular surface inflammation with anterior stromal defect and limbal descemetocele.

Discussion

Rose[1] published a case series of 12 patients (10 females) aged between 77 and 93 presenting to Moorfields Eye Hospital with chronic relapsing purulent conjunctivitis affecting one eye and lasting between 8 and 48 months.

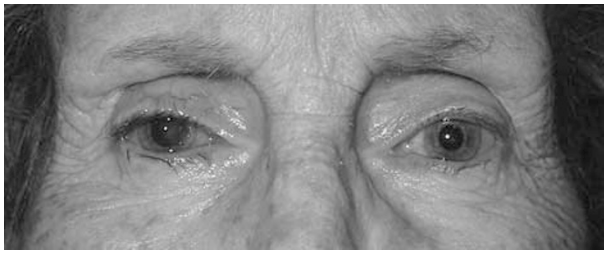

Figure 3 Age-related dehiscence of the levator aponeurosis and orbital fat atrophy.

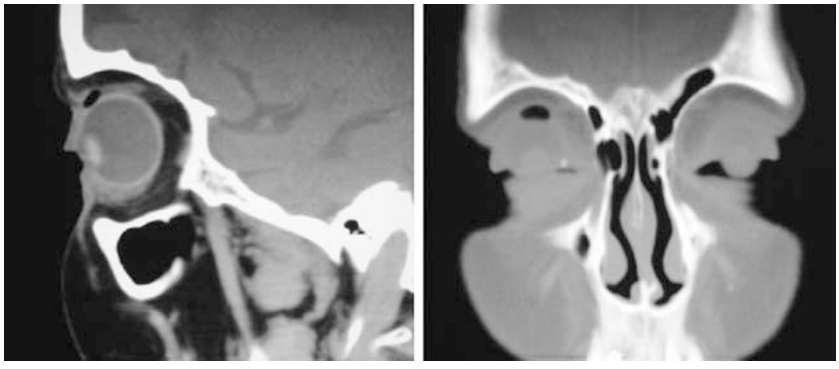

Figure 2 CT scan of the orbits demonstrating air in the right superior conjunctival fornix.

Three had undergone successful DCR before the diagnosis of Giant fornix syndrome was made. Nine had corneal vascularisation and scarring before referral. Five had suffered spontaneous perforation or thinning. As in this case, all had deep upper fornices and associated changes of age-related dehiscence of the levator aponeurosis and universally, *S. aureus* was the inhabitant.

He postulated that gradual deepening of the upper fornix owing to age-related disinsertion of the levator aponeurosis allowed for the accumulation of a bacteria-laden protein coagulum within a capacious upper fornix. Persistent reinoculation of the tear lake by low-grade bacteria from the fornix, severe conjunctivitis from 'toxic' bacteria and the development of a pseudo-membrane, may enhance the coagulum and also exacerbate the ptosis and deepening of the upper fornix. This environment leads to severe ocular surface inflammation.

This case highlights the need to consider a deep upper fornix, present in many of our patients with age-related levator disinsertion and orbital fat atrophy, as the source of a recurrent (often unilateral) purulent discharge. In addition, we suggest that CT imaging is beneficial in excluding frontal sinus fistulae and foreign bodies in such cases, and the presence of air in the deep upper fornix of this patient on CT scan is a new sign of GFS, not described previously.

Reference

1 Rose GE. The giant fornix syndrome: an unrecognised cause of chronic, relapsing, and grossly purulent conjunctivitis. *Ophthalmology* 2004; **111**: 1539–1545.

SJ Turner, V Sharma and PA Hunter

Department of Ophthalmology, Kings College Hospital, London SE5 9RS, UK

Correspondence: SJ Turner,
Tel: +7884497831;
Fax: +2073463738.
E-mail: stove@doctors.org.uk

Eye (2006) **20,** 1481–1483. doi:10.1038/sj.eye.6702370; published online 26 May 2006

Sir,

Third nerve paralysis as a presenting sign of essential thrombocythaemia

Essential thrombocythaemia is a clonal myeloproliferative disease characterised by a sustained platelet count in excess of 600×10^9/L and clinically, by episodes of thrombosis and/or haemorrhage.[1] The thrombotic events primarily affect the microvasculature. Ocular complications are rare and include central retinal vein and central retinal artery occlusion. We report the clinical findings in a patient with essential thrombocythaemia who presented with partial third nerve paralysis; an association not previously described.

Case report

A 74-year-old man presented with a 1-day history of double vision associated with a right-sided frontal headache. He was systemically well and not on any regular medications. He was not a smoker. Both temporal arteries were pulsatile and nontender and the patient had no other symptoms of giant cell arteritis. On ophthalmological examination, the visual acuity was 6/6 in each eye and colour vision was normal. The ocular examination including the pupils and optic discs was entirely normal in both eyes. Evaluation of the extraocular movements showed limitation of adduction, elevation, and infraduction in the right eye. No ptosis was detected. Examination of the other cranial nerves was within normal limits. These findings were consistent with a diagnosis of pupil-sparing partial third nerve paralysis.

At presentation, the patient's blood pressure was raised at 160/100 mmHg. MRI scan of the orbits and brain was completely normal. The ESR, CRP, blood lipid, and blood glucose levels were within normal limits. Further haematologic studies showed neutrophilia (18×10^9/l) and an extremely high platelet count of 1024×10^9/l (normal $150–450 \times 10^9$/L). Bone marrow biopsy was performed, which revealed a hypercellular marrow with atypical megakaryocytes but no marrow fibrosis was noted. Chromosomal analysis was normal thus excluding a diagnosis of chronic myeloid leukaemia. Essential thrombocythaemia was diagnosed and the patient was started on hydroxycarbamide and aspirin. The blood pressure was controlled with medication. Within 2 weeks the platelet count decreased to 315×10^9/l. The diplopia improved rapidly and the patient was essentially symptom free a month after starting treatment. At the end of 5 months of follow-up, his platelet counts remained stable on medication and the third nerve paralysis had almost completely resolved.

Comment

To our knowledge, this is the first report of an association between essential thrombocythaemia and third nerve